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COMMUNICATIONS

PARENCHYMATOUS KERATITIS IN TRYPANOSOMIASIS IN CATTLE AND IN DOGS, AND IN MAN*

BY

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LONDON

IN the course of trypanosomiasis in cattle and in dogs, the result of experimental inoculation, there is frequently developed parenchymatous keratitis. Experimental dogs inoculated with nagana in Zululand included many which became blind. A series of seventeen used in Nyassaland contained eight which went blind (David Bruce). Warrington Yorke in the experimental farm at Runcorn found frequent occurrence of interstitial keratitis not only in dogs, but in horses, donkeys, and goats. Morax, referring to work by Stock, states that, after inoculation into the anterior chamber of dogs and rabbits of infected blood, keratitis develops, with oedema of the cornea, the development of deep vessels and mononuclear infiltration. Abundant trypanosomes were found at the margin of infiltration and at the edge of healthy tissue. The same author states that in the goat the disease is absolutely comparable with syphilitic interstitial keratitis in human beings. In the course of a few weeks the cornea became opaque and gradually whiter and whiter. This was followed

*The illustrations of this paper were shown at a meeting of the Comparative Section of the Royal Society of Medicine on January 5th, 1927, when microscopic sections were displayed.

by a slow clearing of the opacity so that in a few months only a few striae of cicatrization remained. Similar is the condition in the horse. Iritis or irido-cyclitis also occurs with this condition, but not invariably. The trypanosome has been demonstrated in the aqueous and in the iris and ciliary body. Stock describes the appearance in the cornea in this experimental keratitis as an opacity which is porcelain-white in colour and contains deep vessels. He found no organisms in the cornea but a few in the aqueous. Daniels reported in 1911 the development of keratitis in a brown dog affected with the disease. In this case, injections of arsenophenyl-glycin caused a rapid clearance of the greater part of the corneal opacity, which relapsed later after an interval of a few days. The opacity was again abolished by a repetition of the treatment and again recurred. In each case with the reappearance of the interstitial keratitis, trypanosomes were found in the blood. Warrington Yorke described cases of parenchymatous keratitis in some goats and a horse; in some of these cases he found trypanosomes present in the inflamed cornea. Descriptions of the course of the clinical condition in the eye were included. Woods describes in detail his findings in experimental trypanosomiasis keratitis in a dog. de Schweinitz and A. C. Woods, in an experimental investigation on dogs, inoculated the animals intraperitoneally with infected blood, and increased the virulence by successive passages. Ocular signs appeared in from five to twelve days of the time of first discovery of trypanosomes in the blood. The clinical signs were clouding of the cornea, haemorrhage and exudate into the anterior chamber, and severe iritis. Pathological examination revealed thickening and infiltration of the cornea, the formation of new capillaries, and the presence of trypanosomes between the corneal lamellae, stained by alum-iron haematoxylin. There was cell infiltration of the iris, but no trypanosomes were discovered here or in the ciliary body. The ciliary processes were embedded in a fibrinous exudate containing a few organisms.

Dr. Muriel Robertson wrote in 1921 to the author that she had many times seen dogs develop keratitis in acute trypanosomiasis of the Gambiense-Brucei group.

The writer describes below in detail the histological findings in an eye affected with parenchymatous keratitis in a dog infected with the disease. The specimen was from a dog examined by Dr. C. W. Daniels. The writer is indebted to Mr. E. Treacher Collins for permission to examine the eye and to report on the condition, and to Moorfields Eye Hospital for laboratory facilities. The salient points are vascularization of the cornea in all its layers, marked cell infiltration in the peripheral parts and the presence of abundant trypanosomes in the infiltrated area of the cornea. The cellular exudate in the anterior chamber also contains abundance of

poorly stained trypanosomes. The iris and ciliary body show slight infiltration, with abundance of trypanosomes, whilst the ligamentum pectinatum is markedly affected.

Several reports of the occurrence of an interstitial keratitis in men affected with trypanosomiasis have been made (Shirecore five in twenty-nine cases, Thiroux one in four cases), also of irido-cyclitis. Daniels reported a total of forty-two cases of trypanosomiasis in men in Rhodesia, Nigeria, Uganda and the Congo—cases which were examined in England. Among these there were thirteen

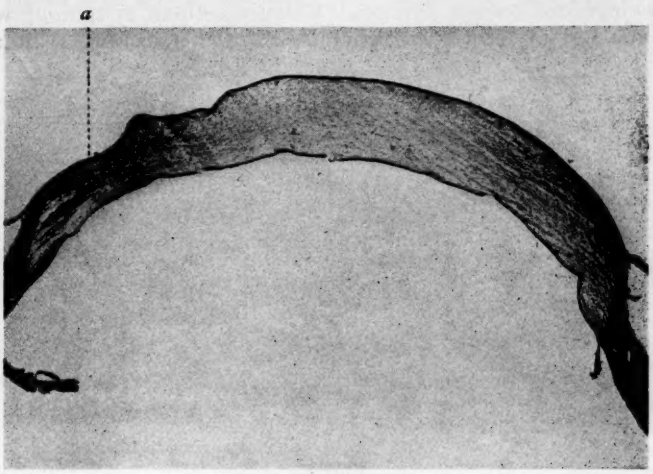

FIG. 1.

Dog. Path. No. 1536 (Moorfields Eye Hospital). ×6. Anterior portion of eye shows peripheral infiltration and vascularization, *a*.

deaths, and fifteen cases with ocular lesions (35.7 per cent.). Daniels emphasizes the importance of the signs which occur in the eye as directing suspicion to a possible trypanosome infection. The affected eye is often painful, the condition always noticeable, and in some cases it has been the first symptom that caused the patient to consult a medical man. The lesions are described as being similar to those in dogs but of a milder form. Daniels noted ciliary congestion, slight keratitis and iritis. One of his cases was shown by Leslie Paton at a meeting of the Ophthalmological Section of the Royal Society of Medicine and is described as having had very slight photophobia and irido-cyclitis, with a rash resembling bruises of the skin of the body, and enlarged glands and slight pyrexia. No reference to pathological examination in man has been found.

Pathological Examination

Dog. Royal London Ophthalmic Hospital, Pathological Specimen No. 1536.

Keratitis in Trypanosomiasis.—Right eye of a black dog. This specimen was given to Mr. E. Treacher Collins in 1911 by the School of Tropical Medicine. The writer acknowledges his gratitude to Mr. Collins for the remaining portion of the specimen. The half eye was treated with gum solution and frozen sections were prepared in the ordinary way. A portion was embedded later in paraffin. Various methods of staining with Giemsa's stain were tried, and also Ehrlich's haematoxylin and

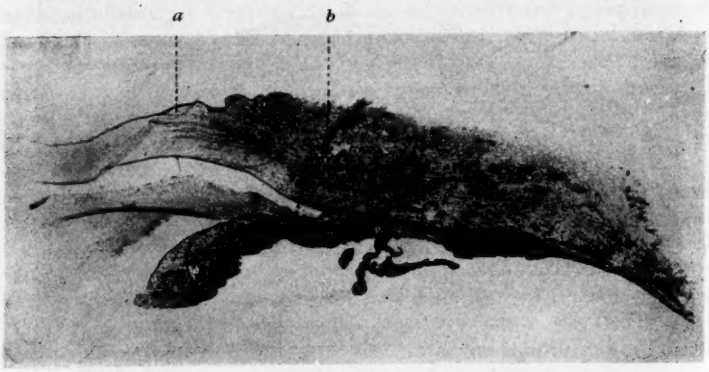

FIG. 2.

Corneo-scleral junction, ×7, iris, and part of anterior chamber; shows peripheral vascularization, *a*, at all depths of the cornea; mass of coagulum in anterior chamber. The portion, *b*, is reproduced at greater magnification in *Fig. 3*.

eosin. The most satisfactory sections were obtained by the latter method of staining in the case of paraffin sections.

Microscopical Examination.—The cornea shows intact epithelium. There is infiltration of the substantia propria, greatest at the periphery and fading off towards the centre from both sides. The infiltration is most marked in the deeper layers of the periphery and is composed of polymorphic cells for the most part, with very diverse forms. There is present a zone, as seen in the sections, of cell infiltration and proliferation which encloses the central deeper parts of the substantia propria. Within this zone can be traced a falling off of the staining power of the corneal cells only in the deepest central layers. This indicates necrosis of this portion of the substantia propria. There are the stained nuclei of a few cells of infiltration

(polymorphic). Peripheral to the zone of infiltration there is a narrow zone of slighter infiltration. Some delicate capillaries penetrate the cornea from the corneo-sclerotic junction. Amongst the vessels is a high degree of infiltration. The capillaries pass inwards beneath the limbus at all depths of the substantia propria, and for a distance

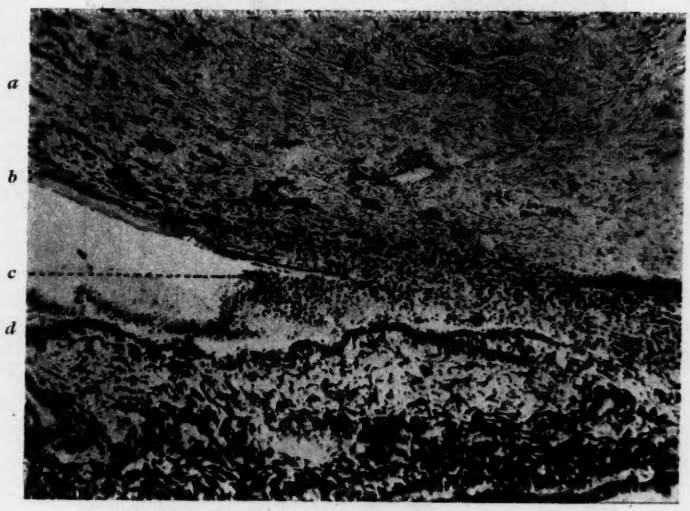

FIG. 3.

× 90, shows a small portion of the above, including the extreme periphery of the angle of the anterior chamber. (*a*) Posterior lamellae of cornea with masses of normal pigment. (*b*) Posterior glass membrane (Descemet) with endothelium on its posterior surface. In front of this, a small amount of cell infiltration is visible. (*c*) Cell exudate in angle of anterior chamber with lighter stained fibrinous material. This cellular exudate is continuous with cellular infiltration of the ligamentum pectinatum. (*d*) Anterior surface of iris, deeply pigmented. The substantia propria in the photograph shows an excess of cells above the normal and some cell infiltration around the vessel near the middle of the photograph.

of about 1.5 mm. mesial to the limbus on each side of the section examined. On one side immediately anterior to Descemet's membrane is a collection of red corpuscles which has the appearance of a haemorrhage, by which the layers of the substantia propria are pushed apart. Trypanosomes are to be seen in various parts of the substantia propria particularly numerous within a short distance of the capillaries. Most of them appear to be in or on the lamellae of the substantia propria,

a few are seen in spaces between the fibres. As many as 15 trypanosomes were counted in a single field in examining with a 1/12th oil immersion objective and No. 4 eyepiece (Zeiss). In another part the trypanosomes were so closely packed as to be uncountable.

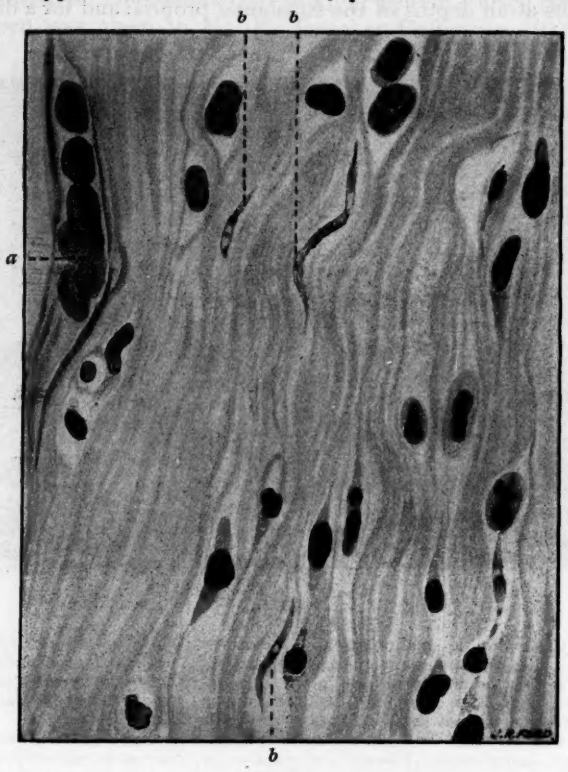

FIG. 4.

Drawing of portion of above under magnification by 1/12 oil immersion objective and No. 4 E.P. (Zeiss). (*a*) Red corpuscles in capillary in the substantia propria. (*b*) Trypanosomes. In a section of the cornea, the whole of a trypanosome is rarely seen in one position of the focussing. It is only by racking up and down that the whole length of the trypanosome is visible. Some trypanosomes are only present in part in sections. Presumably these have been divided by the microtome knife.

The anterior chamber contains masses of inflammatory cells among which are many poorly stained trypanosomes.

Some sections show much cell infiltration of the ligamentum pectinatum, and continuity of this with abundance of inflammatory cells in the anterior chamber and many also in the ciliary body. In

the spaces of the ligamentum pectinatum, numerous trypanosomes can be seen with the oil immersion objective. The iris in one place, near its pupillary border, contains a dense nodule of cells, most of which are polymorphonuclear. Examination of the vitreous cavity

FIG. 5.

× 600. Section of part of vascularized and infiltrated cornea showing trypanosomes, t^1, t^2; t^1 indicates part only of a trypanosome, immediately below which is a complete organism. In the upper left hand quarter of the photograph are very numerous trypanosomes. (Microphotographs by F. Welch.)

on one side at the ora serrata reveals a few inflammatory cells, and among them many trypanosomes.

A fact of practical importance in the examination of the trypanosome in sections of tissue is that this organism appears very considerably smaller than when seen in smear preparations or blood films. In the latter it becomes flattened out and hence appears to be much wider than when fixed in tissue.

Conclusions

In trypanosomiasis in animals of various species, and perhaps also in man, a parenchymatous keratitis is often produced. In some cases this is accompanied by iritis and cyclitis. Pathological examination in animals reveals a severe degree of inflammation of these structures, with marked new formation of vessels in all layers of the cornea and in one case (dog) some necrosis in the centre of the cornea. As far as has been ascertained it bears the closest resemblance clinically and pathologically to distemper keratitis in dogs and to syphilitic interstitial keratitis in man. In the more severe cases of the latter, necrosis of part of the cornea takes place with consequent dense scar formation and very serious impairment of vision. It is probable that pathological examination of late stages of severe cases of distemper in dogs and of trypanosomiasis in various animals would reveal a similar scar formation in the centre of the cornea—as a cause of serious visual impairment. Pathological examination of periodic ophthalmia in horses, of tuberculous keratitis in man and in cats, and of leprous keratitis in man, reveals similarities but also various differences. In distemper in dogs, trypanosomiasis and syphilis in man and animals, the affection of the eye is a local manifestation of a blood infection, the lesions being due in the case at least of trypanosomiasis to the immediate action of the organisms or the effect of their toxins in the vicinity and is an important physical sign of disease.

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A CASE OF SYMPATHETIC OPHTHALMITIS

BY

D. J. Wood

CAPE TOWN

This disease is becoming rare and, in my experience in South Africa, extremely rare. I have seen two cases in white women occurring after operation and after the offending eye had been already excised. The last of these was twenty-five years ago. I have never seen a case in a coloured person, or in a native, despite the numbers of severe injuries which these people get, and where the advice to have the eye excised is often refused.

The present case presents some unusual features, among which one must count its remarkably fortunate result.

The patient was a fair haired girl, aged nine years, who was so unlucky as to contract gonorrhoeal conjunctivitis from a girl with whom she was playing, and who proved to be affected with vaginitis. Both eyes were affected, and when I saw her the right had already got a deep marginal ulcer. This perforated next day, March 21, 1926, leaving a large prolapse.

Two weeks elapsed before the eye was quiet enough to have anything attempted, but then under an anaesthetic I made a large conjunctival flap, removed the prolapsed iris, and stitched the flap over the hole. Unfortunately, as the first stitch was put in the lens capsule gave way, and most of the lens escaped. If I had had permission I should then have excised the eye. By the next week, however, it was clear that the flap was going to hold in place and the eye was quiet. I have more than once saved a pretty hopeless eye by this treatment. The eye remained soft, but this was attributed to the fact that probably a large drainage area had been made. The visible iris was normal in colour, there were no precipitates on the cornea, and, presuming on my immunity from sympathetic disease, I was not anxious.

The patient left hospital on April 22, but as she was leaving my Ward Sister said she thought the left eye had looked pink on the previous day. Ordinary examination disclosed nothing wrong, but to be on the safe side I had the patient brought to me next day and examined her with my slit-lamp. I was distressed to find that there were very fine precipitates on the cornea, cells in the aqueous, and a dewy endothelium. The child was sent back to hospital and the right eye excised on April 24.

While under the anaesthetic 200 c.c. of blood were removed for preparing an auto-serum and a dose of salvarsan was given. Atropine and dionine ointment was begun at once and continued all through. The auto-serum was used as recommended by Guiral

but after the sixth injection of 3 c.c. it became slightly septic and caused a temperature and a local erythema. A second supply was destroyed by overheating.

Meanwhile the pupil had dilated well and the precipitates, which at first increased considerably, grew less. The vitreous was very hazy, but one could see that there was congestion of the retinal veins (V.=6/36). On May 23, after a consultation, sodium salicylate was administered in large doses, as recommended by Mr. Werner. The salvarsan had been kept up once weekly. In spite of my fears, and a few temporary exacerbations, the eye kept surprisingly quiet, very little watery, and nearly free from pain. The lens was covered, except in the centre, with fine pigment deposited from the aqueous.

On June 9, I was cheered by finding that the patient read 6/9; on July 6, vision had gone up to 6/5. I found the aqueous practically cell-free and the precipitates were beginning to show absorption at their edges. This was eleven weeks after the disease was recognized, and such a result exceeded all our hopes. I saw her again in the beginning of November. There were still plentiful traces of old precipitates, but the pigment on the lens had mostly become absorbed, and there were only a few fine fibrinous deposits, probably marking the site of the early adhesions. Though the brunt of the disease fell on the choroid there were at no time visible spots of choroiditis, nor was the tension much diminished.

Where so many lines of treatment were used one cannot assess their values. Salvarsan was used throughout, but most of the improvement occurred during the time when the auto-serum was administered. The early improvement would, however, be the most noticeable in any case. The worst of the trouble was over before the salicylate was started. There can be no doubt that the most important factor was the recognition by the slit-lamp of the disease in its very early stage and the immediate removal of the exciting eye.

The excised eye was hardened in Bouin's fluid and sectioned in celloidin. The sections were stained with various haematoxylin stains, mainly Heidenhain. Pappenheim's stain was disappointing, possibly owing to the fixative.

Considering the fact that the eye had retained perception of light and had been nearly free from redness, the changes found were surprising. No part was quite unaltered while most parts were profoundly altered.

The retina was everywhere detached and the nerve head so much drawn forwards that at first I thought there was papilloedema. I have since found that in cases of detachment associated with inflammation this is common and may be due either to active

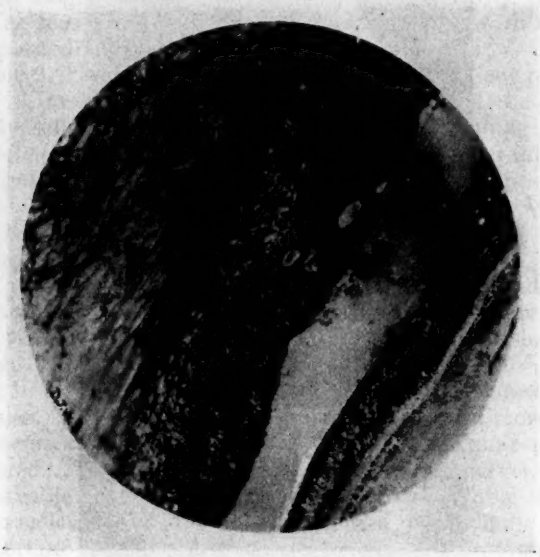
Sympathetic ophthalmitis. Shows the conjunctival flap, the thickened iris, detachment of retina, and pushing forwards of optic nerve.

Section through optic nerve. Choroid spaced out in deeper layers, infiltrated in vascular layers.

pushing forwards of the retina by exudate or to a negative pressure in front.

The retina was unaltered and stained well and sharply by Heidenhain's method. Most of the vessels near the head of the nerve were surrounded by lymphocytes, but not for any distance up the nerve, nor did the changes reach far from the nerve into the retina.

The choroid showed typical changes. The deeper layers were spaced out through their entire length and the stroma pigment

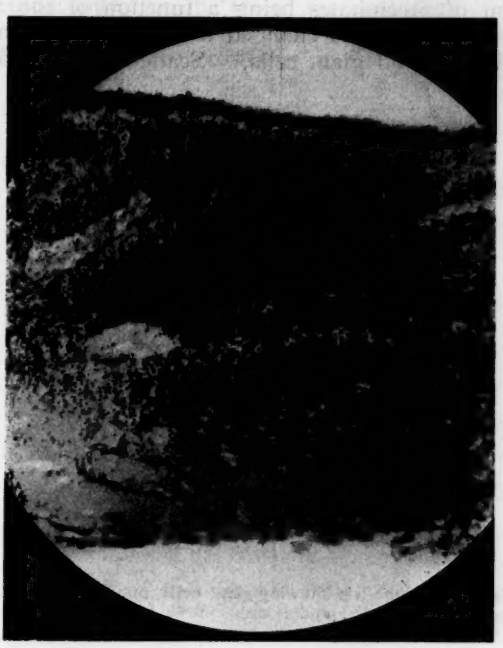


Section of choroid showing nodules of lymphocytes. ×100.

cells were very distinct. This, I think, was the result of the fixative causing contraction in the inner layers.

The inner layers were changed into a thick mass of lymphocytes arranged in confluent nodules. Among them were the nuclei of epithelioid cells and many small giant cells, with oval nuclei from six to a dozen in number. Both in the choroid and ciliary body one could see that the pigmented cells of the stroma, derived from the mesoblast, were undamaged. Their processes were intact and no pigment could be seen scattered outside them. The pigment of these cells is of course in very fine granules without any recognizable shape even with high magnification, whereas in the hexagonal pigment and its forward extension

over the iris the pigment granules are in two forms, large granules more or less rounded which lie near the bases of the cells and are found through the whole uvea, and fine rods which lie in the part of the cells next the rods and cones, and are found more sparingly beyond the retina. In the present case there has been great



Section through a lymphocyte nodule showing intact stroma pigment cells. × 200.

damage done to the pigmented cells derived from the epiblast, especially in the front part of the eye.

The deposits of lymphocytes thinned off as one traced the uvea forwards, but continued without a break, becoming again much denser and thicker in the ciliary body and iris.

The angle of the anterior chamber was destroyed on one side by the prolapse, but was fairly normal though very narrow on the other.

The iris was surprisingly altered. During life the colour had been but little changed, though the surface was irregular. Sections showed that the tissue was greatly thickened, and that the pigment layers forming the posterior surface were completely

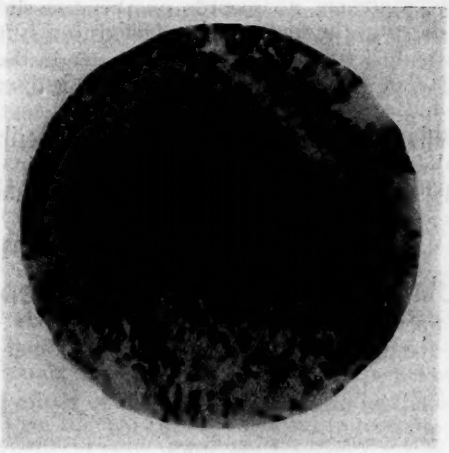
broken up and the pigment scattered. At only one spot near the intact angle of the anterior chamber was there a small continuous layer of pigment. The iris was adherent in part to Descemet's membrane; on the rest of the membrane no precipitates could be seen, agreeing with the naked eye observation. I think this was due to the shallowness of the anterior chamber, *ceteris paribus*, the amount of precipitates being a function of the amount of aqueous. The iris was changed into a mass of lymphocytes, epithelioid cells, and giant cells. Scattered all through were

Section through iris showing giant cells, nuclei of epithelioid cells, plasma cells, and lymphocytes. Pigment granules in giant cells and scattered elsewhere. × 300.

remains of pigment, much of it extra-cellular and in large granules, but some ingested by the epithelioid cells and outlining their bodies. There were also many plasma cells. These never occurred singly but were in groups related to a blood-vessel. This was very evident near the surface of the iris where there were many new vessels, some not much more than a wall of endothelium. With Heidenhain's staining many of the lymphocytes showed an arrangement of their chromatin approaching very closely to that of the plasma cells.

In the ciliary region the damage to the pigment cells was also very great. The pigmented layer was thinned and there was loose pigment visible everywhere. Here again the granules were nearly all of the rounded type and on the whole seemed larger than those which occur normally. The unpigmented epithelium seemed to be

unusually prominent, but as was noted by Dr. McIlroy in her interesting paper (*Roy. Lond. Ophthal. Hosp. Reps.*, Vol. XVII), the cells seemed to be surrounded by an exudate which prevented a sharp view. Nucleoli were usually present, but the clear chromatin-marking seen in the epithelioid cells was either finer or less visible. Nowhere could these cells nor the nuclei of the pigmented epithelium be traced far away from their normal sites. Giant cells were less numerous than in the iris but much larger. They lay for the most part in the ridges of the ciliary



Giant cells from conjunctival flap. × 300.

processes and contained often up to forty nuclei. The cell body was always shrunken from its surroundings, probably by the fixation effect, and pigment could always be found in it and in smaller granules than the pigment outside.

The interior of the ciliary processes contained lymphocytes, plasma cells, and numbers of epithelioid cells, probably derived from the stroma. Epithelioid cells were abundant in the outer part of the ciliary body away from the pigment. Most had elongated nuclei resembling those of the endothelial cells of the blood-vessels, but others had large oval nuclei and these I think came from the stroma cells. My sections did not indicate that epithelioid cells came either from the mesoblastic chromatophores or from the cells belonging to the anterior part of the retina.

Nowhere in any section could I find polymorphonuclear leucocytes, except in the blood-vessels, where their occurrence was curiously variable. Some vessels had their blood contents studded

with leucocytes while in others there were few or none. This is probably not related to the process at work in the particular case.

Another curious and disquieting finding was that not only did my conjunctival flap contain lymphocytes in nodules, but many sections showed large giant cells, so wide-spread had been the effect of the disease.

Since I have perforce been made familiar with the appearances found in sympathetic disease I have discovered similar changes in sections of an eye excised as probably dangerous last year. Unfortunately, there are no records on the case-sheet as to the history, etc., beyond the fact that it referred to a European male. Evidently I was lucky enough to excise the eye in time to prevent disease in the other eye, as no case of sympathetic disease has since been recorded.

It is probable that the reason for the case described being so insidious and free from redness, etc., was that it was a pure case of sympathetic disease without any endophthalmitis in the exciting eye.

CALCIUM DEFICIENCY IN THE BLOOD WITH REFERENCE TO SPRING CATARRH AND MALIGNANT MYOPIA

BY

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FOR a good number of years I have been aware that some ocular conditions are associated with or caused by a deficiency in the amount of calcium in the blood, and I have treated cases empirically with good results, though I had not the means of ascertaining whether such deficiency really existed. I have now for some time been able to have this done through the kindness of Dr. L. Bosman, of the bio-chemistry department of the University of Cape Town, and it has been interesting to find that my expectations have been realised in all the cases where I was able to get the patient at a sufficiently early age.

The first cases to which my attention was drawn were sufferers from spring catarrh, and my early experiments were made with "afenil," a calcium chloride urea made by Knoll. I found that where the patients were young and the disease affected the pericorneal zone a cure could be effected in as little as three weeks by the giving of a 10 c.c. ampoule of afenil intravenously (one grm.) thrice in a week. The thickenings faded away and left only a narrow

opacity near the edge of the cornea. Unfortunately the cure was not permanent, but that it was related to the treatment was shown by cases where repeated treatments produced disappearance of the thickening, in one case three times. The effect on spring catarrh affecting the lids was less satisfactory, but in these cases one had to deal with a grosser manifestation of the disease, where there was much epithelial hypertrophy, often extending deeply in the follicles, so as sometimes almost to be mistaken under the microscope for epithelioma. The follicle consists of a core of fibrous tissue with few cells, an intermediate area where numbers of plasma cells are found, with the endothelium of blood channels, and on the outside a thickened layer of epithelium usually showing some degenerative change. One was, however, able to convince oneself that in these cases there was also a real relation between the condition and a calcium deficiency, as treatment shortened the duration considerably.

For about a year I have had all cases examined as to the calcium content of the blood and in every case where the child was young enough a notable defect was revealed. The last three cases showed values of 9.3 mg., 8.9 mg., and 8.5 mg. per 100 c.c. of blood, in place of the normal for such ages, *viz.* 11 mg. By the time, however, that a child is into its 'teens the calcium deficiency appears to be made good.

For some years, however, one had learned that calcium administration alone is not much good, and that it should be combined with parathyroid extract. I confess to have had a disbelief in the administration of tiny doses of the parathyroid gland in the form in which patients could get it. This has been found to be quite erroneous, as the small amount of one tenth of a grain twice or thrice daily, without any other medicament will usually raise the calcium content to normal, or above it, in a few weeks. In most cases calcium lactate or kalzana has been administered as well.

As regards other treatment, occlusion of the eye is of great value. In cases of spring catarrh confined to the pericorneal site, and occurring in young children, occlusion, combined with parathyroid, will cause the disappearance of the swelling in two or three weeks, and if the blood calcium be watched and kept normal there should be no recurrence. Simple saline or boric acid irrigation is used to combat germ proliferation. In the lid cases the duration of the disease is shortened, the period of discomfort is reduced to months in place of years, though the nodules require a much longer time to vanish. I am unable to suggest the *modus operandi* of the calcium deficiency on the tissues. The trouble occurs at the period when the bones are beginning to make a great demand for lime salts, and as bones are most important from a developmental point of view they get their share and other tissues may be stinted. The possibility is that there is a want of endocrine balance between

the glands such as the ovaries, whose hormone I am told by Dr. Bosman, permits the release of calcium from the blood, and that of the parathyroids which tends towards its retention. Spring catarrh might be defined as a reaction to climatic conditions—dryness, heat and dust—in young children where there is a deficient calcium content in the blood.

There is a difference in prognosis between the cases where the lids alone are affected in spring catarrh, and those where the disease is solely or largely pericorneal. The former are slower to get well, but do so entirely, so that in after years there is no visible trace left. In the latter, where the pericorneal change is considerable, there is a risk of the development of conical cornea. I am inclined to think that this cause of kerato-conus produces its effect rather quickly, but the final result is similar to what we ordinarily know as conical cornea, except that linear opacities are left at the margin of the cornea which last for a long period. I have recently seen an old spring catarrh patient where a plus 9.0 D. cyl. crossed with a minus 6.0 D. cyl. gave vision of 6/36, a condition comparable with ordinary conical cornea results. Fortunately the other eye was less affected. If conical cornea may develop as part of a case of spring catarrh, an association beyond doubt, is it not very probable that the same aetiological factor is present in conical cornea which develops without it? The age incidence is the same, and the period of cessation of rapid advance is similar. I have, moreover, just seen a case of very early conical cornea. The patient had been complaining of difficulties in vision for some time, and I got the additional information that she had been vaguely ill, had not menstruated for over six weeks, and that previously her periods had been irregular. Her eyes showed the characteristic retinoscopic appearances seen in kerato-conus of low degree, and her choice of lenses was quite different from my estimate. With weak convex cylinders she got 6/6 nearly. Her calcium content proved to be the lowest I have found so far, viz., 7.2 mg. per 100 c.c.

In view of the fact that there has been no menstrual period this is very low, and one would expect that, if menstruation occurs soon, the calcium level would fall still lower. It will be interesting to watch this early case. I am of opinion that in all such cases there has been a general flabbiness and want of tone which suggests a deficiency of something. I do not think I have ever seen a case of conical cornea in a patient who was short in stature, but I have seen too few to say that the reverse is true, *viz.*, that it occurs in patients whose bones have grown too fast. If we grant that the cornea has not got toughened up or has lost some of its toughness, we should expect that this, combined with a peripheral source of nutrition, would result in the curve with which we are familiar in kerato-conus.

Myopia

If the cornea tends to give way in calcium deficiency, it is no long step to assume that the sclerotic may also give way, and that this may be associated with the advent of myopia. The condition is sufficiently common in youth for plenty of material to be available, and I have obtained proof in cases of rapidly advancing myopia in young children that the blood shows a calcium deficiency. Myopia is, however, a complex condition, and more than one factor is present. I have had several cases of high myopia in children of three years of age, and I have seen others, which though they were not recognised so young, had probably been in existence for a long time. The unexpected result in following up the cases was that in spite of one's fear the myopia did not increase, and the ophthalmoscopic picture was that of a fairly normal eye. For instance, many years ago a child of two years and eight months was brought to me where there was as much as 13.0 D. of myopia. The greatest care was taken of the case, and I used to think that I had been the means of saving her eyesight. She saw Mr. W. Lang when she was half grown, and I have seen her lately, a tall young lady of more than twenty with vision of 6/4 in each eye, no myopic changes and no increase of her original myopia. I have seen another case slightly younger, with minus 14.0 D. sph. This case I saw till she was seven without there being any change. It is not possible to put these cases into any category except congenital myopia, and I am satisfied that every now and then nature makes a myopic eye. The next cause is that the eyeball may grow normally beyond its correct dimensions. I have more than once seen a large family whose eyes when they were very young were nearly emmetropic and where one after the other have become myopic to a moderate degree. One such family, all girls, are now young women and are all myopic to about five dioptres. Similarly another family, now all grown up, came to me when young with from two to four dioptres of hypermetropia. They are now all within a fraction of emmetropia. Such cases change without one being in the least able to control them, but though there may be slight myopic changes in the fundus they never get seriously short-sighted.

In very sharp contrast with these are the cases where a child originally normal becomes rapidly myopic in the early years of life. My average record of these cases is that in spite of all care they advance up to adolescence and usually long past it. A full correction and apparently good health, with a proper reading range merely slow the advance but do not arrest it. Here we must admit that the sclerotic gives way, and we see the results of this reflected in the myopic character of the fundus, the thinned vitreous, and the danger of detachment. The sclerotic gives way as a result of some

primary cause, and having become thinner, it may go on yielding even if the primary cause is no longer operative. Whatever may be said of the effect of eye muscle pressure, or of the effect of looking downwards at near work, one cannot class these as primary causes. If, however, we find an underlying factor which comes into play when growth begins to be rapid, and which produces a weakening of the sclerotic, then the difficulties in connection with the onset of myopia are largely solved. If calcium deficiency will result in bulging of the anterior part of the eyeball, why should it not do the same to the sclerotic. That it does so I think I have now proved, as in every case of myopia coming on quickly, and progressing I have found a calcium deficiency if the case was examined early enough.

The following are typical cases :—

Sheila R., aged 9 years, a thin tall child, just discovered to have defective vision. Right eye − 5 D. sph., left − 1.0 D. sph. with −1.5 D. cyl. Calcium content found to be only 7.5 mg. per 100 c.c. This case went up to 12 mg. in six weeks on parathyroid treatment only.

Richard R., a child with hereditary myopia in the family, was brought for blepharitis. Aged 9 years and 6 months. Each eye had − 2 D. sph. with − 0.5 D. cyl. Calcium content 8.5 mg.

Willy K., aged 4 years, rather neglected. Right eye − 7 D. sph. with 1 D. cyl., left eye − 6 D. sph. with 1.5 D. cyl. Calcium content 8.5 mg.

Rosie L., aged 9 years, hereditary myopia. Right eye − 6 D. sph. with − 3 D. cyl., left eye − 5 D. sph. with − 3 D. cyl. Calcium content 9.5 mg. This went up to 12 mg. in a few months, but on stopping the treatment it dropped to 10 mg. No increase in myopia in the time.

Bernard F., aged 9 years. Six months ago he was first seen, but his blood could not be examined. Since then the right eye has gone up from −1.0 D. sph. to − 3.5 D. sph. and the left eye from −6.0 D. sph. to − 8.5 D. sph. Calcium content is 9.8 mg.

Doreen B., aged 7 years. Right eye − 15.0 D. sph., left eye − 14 D. sph. with − 1 D. cyl. Calcium content 8.9 mg. This case had also old disseminated choroiditis in each eye.

Harry B., aged 6 years. Brought for blepharitis. Right eye − 4.0 D. cyl., left eye + 1 D. sph. with − 3 D. cyl. Calcium content 8.0 mg.

In only one case a different result was obtained.

Sidney H., aged 12 years. Each eye − 15 D. The child was extremely small for his age, and was a seven months' baby. Vision was only 6/18, but there was hardly any myopic change in the fundi. Calcium content 13 mg. Evidently we were either too late, and the demand for calcium by his bones had been small, or it may have

been a case of congenital myopia. All that I could get from his mother was that he had always been ill. This case can hardly be considered an exception to the rule.

I have records of a number of other cases but those quoted will suffice.

In all up to the age of 12 years, a calcium deficiency has been found. After that age the calcium content appears to become normal.

South Africa is a very large country and the difficulty of seeing cases at regular intervals is likely in the future to prevent me from seeing the effects of treatment. It would therefore seem desirable not to wait, but to give those in large centres the chance of testing my views and watching the results. It will be in any case a matter of years to discover if we can prevent the further advance of myopia in early cases.

It would seem obvious that cases of congenital myopia, and growth myopia will be for ever beyond other than optical treatment. If, however, my observations are confirmed generally, then it would seem to be a reasonable hope that cases of malignant myopia with its disastrous results should be preventable. An early blood examination where there is known to be a hereditary tendency to high myopia should reveal the advent of danger, and treatment should prove an effective prophylactic.

I hope to be able to record the results of treatment as time passes.

Note by L. P. Bosman, D.Sc.

It is an established fact that administration of parathyroid gland, not necessarily in conjunction with calcium salts, causes an increase in the calcium content of the blood. A few cases have been cited in the earlier part of this communication having relation to myopia, where the blood calcium went up from deficiency to normal in a short period. As an example in other complaints I may quote the following: Mrs. W., a young lady, aged 24 years, suffering from severe headaches, was found to have a calcium level of only 7.5 mg. Placed on one tenth grain of parathyroid for a month her headaches disappeared, and her calcium content was found to be 11.9 mg. The treatment was stopped and in another month she had dropped to 9.8 mg., and her headaches were as bad as ever. With renewed treatment they again disappeared and the calcium level went up again to 12.0 mg. per 100 c.c.

Mr. L., aged 42 years, suffering from ununited fracture. Calcium content only 6 mg. Treated with parathyroid. In a month the calcium level was 12 mg. Treatment discontinued for two months produced fall to 7.5 mg. Renewed treatment restored it to 11 mg.

The method of estimation is a modification of Tisdall and Kramer. About 7 c.c. of blood are drawn from an arm vein and set aside

without any coagulant. The serum separates out well in twenty-four hours.

Two c.c. of the serum are transferred to a centrifuge tube, and to it are added 2 c.c. distilled water, a few drops of 2 per cent. ammonia, and 1 c.c. of a saturated solution of ammonium oxalate. The contents are thoroughly mixed by rotation and set aside for at least 45 minutes. The tube and its contents are next centrifuged at 2,000 revolutions for three minutes. This results in the calcium oxalate produced in the above reactions being closely and firmly packed at the bottom of the tube. The supernatant fluid is carefully poured off and the tube allowed to drain by inverting it over dry filter paper for five minutes. Two c.c. of 2 per cent. ammonia are now added and the tube again centrifuged and drained as before. Two c.c. of normal sulphuric acid are now added and the tube placed in a beaker of water is now heated to about 80° Cent. The contents are then titrated with N/100 potassium permanganate solution in a micro-burette till a faint pink tinge is left. The solution must be freshly prepared for each new set of estimations. One c.c. of N/100 solution of potassium permanganate corresponds to 10 mg. of calcium.

A CASE OF SYMPATHETIC INFLAMMATION ASSOCIATED WITH INTRAOCULAR SARCOMA

BY

T. HARRISON BUTLER

BIRMINGHAM

with a pathological note by

J. JAMESON EVANS

BIRMINGHAM

CASES of sympathetic inflammation following an intraocular sarcoma in the fellow eye have been reported by Fuchs, Meller, and others, but must be of extreme rarity. The following example of this complication seems therefore worthy of record.

Miss H., aged 20 years, came to the Coventry Hospital on December 8, 1924, and was found to be suffering from severe irido-cyclitis in both eyes. She gave the history that the right eye had been inflamed for five weeks, the left for six weeks. She was quite positive that the left eye began to give trouble before the right. A month previously she had facial paralysis on the left side but this had completely disappeared when she was first seen. There was no history of any headache or vomiting. There had been slight

vertigo but no tinnitus. She had been slightly deaf for as long as she could remember. Three years ago her tonsils and adenoids were removed. She had been treated by her doctor with large doses of iodide of potassium. The mother suffers from myxoedema but has no symptoms or signs of syphilis.

Condition when first seen.—V.R. Fingers at one foot, not improved with glasses. V.L. 1/60. Not improved.

Both eyes show ciliary injection, the irides are muddy, and there are several posterior synechiae. The posterior surfaces of both corneae are thickly covered with "mutton-fat" precipitates. The tension is normal. The right eye is painful and both are tender. The Wassermann reaction is strongly positive. The general appearance of the patient is healthy, and there is nothing to suggest syphilis, but because of the positive Wassermann reaction the patient was sent to the venereal department, and received a course of injections with novo-arsenobillon and was treated with mercury. She was ordered atropine drops and hot applications.

June 15, 1926. The patient has not been seen in the eye department for eighteen months and has never used her atropine. There is now a complete annular synechia with iris bombé on each side and the tension is raised. No precipitates.

July 1, 1926. Tension; right by Schiötz tonometer, 16; left 13.

September 14, 1926. V.R. corrected 6/24. V.L. light perception only.

September 30, 1926. Schiötz, tension, 36.36. Examination with the slit-lamp showed no cells in the aqueous. Chamber very shallow and iris bombé.

October 5, 1926. Gelatinous nodules seen on the pupillary margin of the left iris. Pellucid type of precipitates each eye.

October 19, 1926. Schiötz, 35 and 40.

October 26, 1926. Circum-corneal injection both eyes. Keratic precipitates.

November 1, 1926. Schiötz, 37 and 37.

November 2, 1926. *Iridectomy, left eye.*

November 16, 1926. Schiötz, 44 and 6. The vision of the left eye has improved. Large hyphaema, which is gradually absorbing.

November 22, 1926. *Iridectomy, right eye.*

December 7, 1926. Schiötz, 7 and 12.

December 23, 1926. Hyphaema right eye, that in the left has disappeared. Lenticular opacity both eyes, no red reflex seen. V.R., 1/60 V.L., hand reflex.

January 4, 1927. No hyphaema. The crypts in left iris are greatly enlarged and are seen with the slit-lamp to be filled with a gelatinous material, in fact the whole of the deeper mesenteric layer appears to

be changed into this type of tissue. The coloboma has closed up leaving a small drawn up pupil.

The Wassermann reaction is negative.

February 22, 1927. The left eye, being hard and very painful, *was removed.* On section a white disc-shaped tumour was seen at the posterior pole measuring six millimetres in diameter. This was cut by Dr. Wright, pathologist to the Coventry Hospital, and he reported that it was a sarcoma.

Pathological Note by J. Jameson Evans

Cornea.—Transparent with pericorneal engorgement and some peripheral oedema of the corneal stroma.

Endothelium intact but shows blood cells in the region of the corneo-iridic angle. Spaces of Fontana crowded with blood cells and inflammatory cells.

Iris.—Vessels congested, infiltration of stroma with round cells, chiefly polymorphs. Patch of pigmented uveal cells deposited on the anterior surface of the iris. Pigmented epithelium swollen and desquamating.

Ciliary body.—Congested vessels. Depigmentation and desquamation of epithelium, Some blood round pigmented cells and in the fibres of the suspensory ligament.

Retina.—Vessels engorged. Some haemorrhages and oedema. Detachment in region of the ora serrata. Pigment epithelium depigmented over the major portion of the fundus, and detached by an albuminous exudate in the equatorial region.

Choroid.—Intense venous congestion with some cellular infiltration, not lymphocytic, in posterior region up to the equator.

Sessile growth.—At the posterior pole consisting of mixed cells deeply pigmented, and largely spindle-shaped. Some haemorrhages. No evidence of perforation of the sclera.

Further history.—The removal of the left eye was followed by a gradual but very definite improvement in the condition of the right eye. The globe lost its injection and no pain was experienced. The deposit of precipitates diminished and apparently no new ones formed. Vision was very poor, the patient could get about in her own home but not out of doors. The coloboma was drawn together and filled with an organized exudate. In November, 1926, the eye again became painful and it was found that the tension had risen to 50 Schiötz units. On November 16, 1926, *a large iridectomy was made horizontally outwards.* This was followed by considerable haemorrhage which rapidly absorbed. This operation was most

satisfactory in its effects. There was no inflammatory reaction and the coloboma remained free from exudate. The tension sank to normal and the patient was able to count fingers and to get about well even in places that she did not know.

Comments.—The presence of an intraocular growth was not suspected until the eye was bisected. There would seem to be little doubt that the severe irido-cyclitis was due to toxins liberated by the necrosing action of the sarcoma. The rapid improvement which took place in the sympathizing eye after the removal of the exciting eye strongly supports the idea that the inflammation was sympathetic in type. On the other hand the true Fuchs inflammation was not present nor were there any cells in the aqueous of either eye. It is obvious, therefore, that the case was not one of true sympathetic ophthalmitis, although it was a sympathetic inflammation.

The eye eventually became perfectly white, and the astonishing acuity of 6/12 was gained. The patient has now returned to her work as an assistant in a draper's shop.

CASE OF EMPHYSEMA OF THE CONJUNCTIVA

BY

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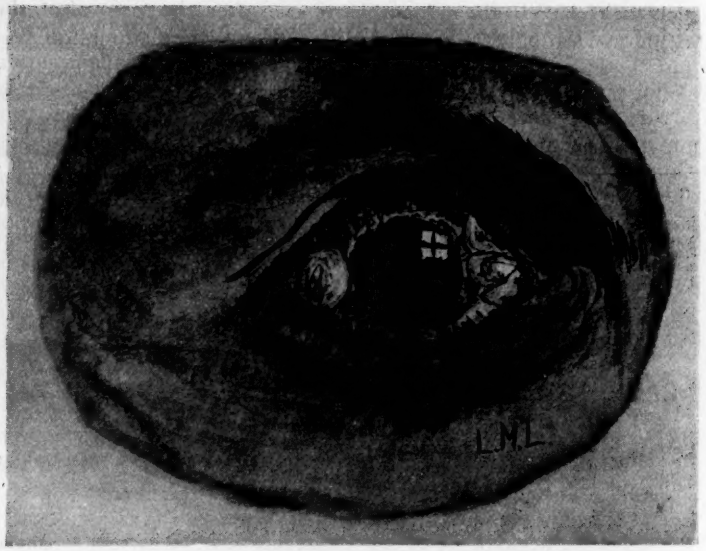
THE following accident is of such unusual occurrence as to merit special attention.

On June 5, 1926, W.B., male, aged 40 years, reported at the Eye Department of the Royal Infirmary, Edinburgh, and gave the following history.

Patient stated that he was employed as an engraver in a granite yard and that he worked with a pneumatic driller. The air which drives these pneumatic drills is compressed by an engine, led through the yard in large iron pipes and smaller rubber tubes and when emerging from the nozzle of the tube has a pressure of 70 lbs. per square inch. It is the custom of the men when they have finished work to blow the dust out of their clothes by means of the compressed air. On the date in question, W.B. took hold of the flexible rubber tube about two feet from its free end and turned on the air, the pressure of which caused the tube to fly backward and pass in close proximity to the face actually touching the skin over the right maxilla. The eye received the full force of the 70 lbs. pressure.

On examination the conjunctiva was raised in several large and very many small bullae which were filled with air and presented an appearance somewhat similar to chemosis of conjunctiva being, however, more loculated. On palpation the typical crackling feeling of emphysema was experienced. The conjunctiva had not much injection and there was no obvious tear to be seen. The pupil was slightly dilated—fundus normal— lens clear—no refractive error



and vision 6/9. The vision in the left eye was 6/4. The accompanying photograph of an original drawing shows the condition on the afternoon of the same day.

On June 7, 1926, there was considerably less air present.

On June 8, 1926, there were only a few air-bells above and to the temporal side. Examination in dark room showed no abnormality and vision on this day was 6/4 the same as the other eye.

Patient was last seen on June 11, 1926, when there was no trace of the accident left save a very slight conjunctival injection. He was then allowed to return to work.

I have not been able to find a report of a similar case in the literature.

I am indebted to Dr. J. V. Paterson, Eye Department, Royal Infirmary of Edinburgh, for permission to publish these notes of the case.

ANNOTATION

The Ophthalmic Benefit and Sight-Testing Opticians

In the oft quoted saying, "the exception proves the rule," the word prove has the same meaning as in the parable in which a man excused himself from attending a great supper because he had bought five yoke of oxen and must go to prove them, that is to test them, so an exception tests a rule.

As a rule medical men who have experience of eye disease regard the practice of sight-testing opticians as fraught with danger. Adrian Caddy, M.D., F.R.C.S., is, however, not in agreement with this rule, and his views, which he has expressed in an outspoken letter to *The Lancet*, afford a test as to its validity.

Dealing with the subject of the administration of ophthalmic benefit he says, "that in the majority of cases the services of an ophthalmic surgeon are unnecessary and that an optician can do all that is needful;" also that he thinks "the *real damage* that can be done to the eyes by unrestricted prescribing by opticians is extremely slight, practically the only risk being that a case of rise of tension in the eye from whatever cause might go undetected."

It will be well here to contrast these exceptional views of Mr. Caddy's with some extracts from the considered Report of the Council of British Ophthalmologists on "Sight-testing by Opticians," published in this Journal in December, 1922. It says:—

"The treatment of headaches, associated with errors of refraction demands not only the use of glasses, but also constitutional treatment, which can only be determined by a medical man. In some cases of defects of the muscular balance of the eyes, the use of prisms is necessary. The correct estimation of these and a complete investigation of the case are most difficult problems, since some of these muscular defects are among the earliest signs of serious diseases of the nervous system.

"Changes in the refraction of the eyes are often produced in the early stage of eye disease. For example, myopia (short sight) often appears in the early stages of cataract, or of chronic glaucoma, a disease which untreated inevitably leads to blindness. Hypermetropia (long-sight) may be met with in the early stages of a malignant tumour. In such cases glasses may bring visual acuity up to a normal standard, but an optician will fail to recognize the disease which will thus be left to run its course.

"Optical defects and disease constantly overlap; the former cannot be differentiated with certainty from the latter, nor the latter definitely excluded by any but a medical practitioner who has had special training and experience."

Mr. Caddy suggests that a patient requiring "ophthalmic benefit" should in the first place be sent by his panel practitioner to an optician, and that only those cases in which the optician is unable to obtain normal vision with glasses should be sent on to an ophthalmic surgeon. It is, however, stated in the Council of British Ophthalmologists' report, that "disease very serious to the eye, or threatening even life, may occur in an eye with perfect acuity of central vision."

Mr. Caddy criticises the way in which the "ophthalmic panel" has been constituted. He says "The ophthalmic panel consists of a list of names and addresses of doctors and nothing more." Apparently he is unaware of the conditions which are required before a practitioner can have his name entered on this panel, and which were drawn up by a joint committee of the Council of British Ophthalmologists and the British Medical Association. They are published in Vol. VIII, p. 591 of this Journal.

We regret to hear that the British Medical Association is showing some reluctance to the reconstitution of this joint committee which did such useful work while it lasted. Combined action by all those interested would be particularly useful just now in connection with a Bill which has been printed and may shortly come before Parliament, entitled the "Optical Practitioners (Registration)." This Bill in its preamble states that it is designed to "secure the registration of optical practitioners and to regulate the practice of sight-testing, and for purposes incidental thereto."

One of its clauses lays down that, "Any registered practitioner shall be entitled to practice optometry, and to give certificates relating to visual acuity or visual defects, which shall be recognised as admissible by any local or central authority or government department in any part of Great Britain." "Optometry" it defines as "the practice of optometry, refraction, or sight-testing, or the examination of the human eye for the purpose of ascertaining any departure from the normal state of vision, measuring its functional powers and adapting or prescribing mechanical means for the aid thereof."

If this bill became law it would mean that Government departments and other public bodies would have to accept from advertising opticians whose business it is to sell spectacles, and who have received no medical training, certificates as to the presence or absence of eye disease. To such a proposal it is suggested that the General Medical Council should give its sanction by appointing one of its members to represent it on the "Optical Practitioners Board of Control."

The Minister of Health has appointed a Departmental Committee to consider the Bill, the members of which are as follow: Mr. F. B. Merriman, O.B.E., K.C., M.P. (chairman); Mr. O. Aves;

Mr. W. B. Barker; Mr. H. B. Brackenbury, M.R.C.S.; Mr. L. G. Brock, C.B.; Mr. E. Treacher Collins, F.R.C.S.; Mrs. W. L. Courtney, O.B.E.; Lord Cozens-Hardy; Mr. R. J. Davies, M.P.; Mr. H. L. F. Fraser, M.A., LL.B.; Dr. C. O. Hawthorne, M.D., F.R.C.P.; Mr. G. E. Houghton, and Sir Henry Keith.

ABSTRACTS

I.—GLAUCOMA

(1) **Derby, Dr. G. S., Waite, Dr J. H., and Kirk, E. B. (Boston). —The light sense in early glaucoma, particularly the achromatoscopic threshold at the macula.** *Trans. Ophthal. Soc. U.K.*, Vol. XLV, Pt. i, 1925.

(1) **Derby, Waite,** and **Kirk** in this paper describe their methods and apparatus for accurate and delicate testing of the light minimum and difference sense. For details of these the original, which is adequately illustrated, must be consulted. The authors begin by plotting out graphs under the standard conditions for 100 normal eyes, discussing the nature of the curves obtained, and comparing them with those obtained from glaucoma patients and from glaucoma suspects. Their conclusions are as follows:

"1. Opposed to the beliefs expressed in the standard texts, we believe with Hecht that, properly measured in the normal eye, dark adaptation proceeds at a precipitous rate for several minutes after the onset of darkness, and that it reaches almost its maximum in ten minutes.

2. For uniformity of results we believe that pre-exposure of the tested eye for five to ten minutes to a known intensity of light is a necessary procedure.

3. We believe that standardization of the pupil is a *sine qua non* for an accurate study of the light-sense, and we offer pilocarpine fixation as the method of choice over all other methods.

4. With the low intensities at which we worked we have been unable to find any significant alterations in the light difference (L.D.) in glaucoma, either in early or established glaucoma.

5. In established glaucoma, light minimum (L.M.) is regularly altered in two ways: first the rate of dark adaptation is retarded, and second the ultimate L.M. is raised.

6. In glaucoma suspects before there is any clinical evidence of the disease, our results show an abnormal alteration in the light minimum, manifested as a retarded adaptation rate, and as an increased ultimate threshold. In brief our results suggest that the light sense in early glaucoma is affected before there is any sustained

increase in intraocular tension, any cupping of the disc, or any visual field changes characteristic of glaucoma. The nature of this disturbance in the light-sense appears to be twofold; first a tardiness in dark adaptation, and second, in the dark-adapted eye, a dulling of sensitivity to dim light.

7. Finally we wish to stress the fact that the results which we present must only be regarded as suggestive until further research on a much larger series of cases has confirmed them."

A useful bibliography accompanies the paper.

E.E.H.

(2) **Derby, Dr. G. S., Waite, Dr. J. H., and Kirk, E. B. (Boston). Further studies in the light sense in early glaucoma.** *Trans. Amer. Ophthal. Soc.*, Vol. XXIV, 1926.

(2) This paper is a continuation of the work published in the *Trans. Ophthal. Soc. U.K.*, 1925 (abstracted above) and contains a number of additional graphs confirming the previous results. **Derby, Waite,** and **Kirk** consider that they are justified in concluding that examination of the light minimum sense is of real value in making the diagnosis of early glaucoma. They further suggest that some simple apparatus, such as the glasses described by Tscherning may be found to give a perfectly satisfactory result, and will make the light minimum sense examination possible in the ordinary consulting room.

E.E.H.

(3) **Knapp, Dr. A. (New York).—Observations on glaucoma in Morgagnian cataract.** *Trans. Amer. Ophthal. Soc.*, Vol. XXIV, 1926.

(3) In this paper **Knapp** records two cases of Morgagnian cataract accompanied by raised intraocular pressure. In the first case the pressure was raised before operation (T. 55 mm.), but improved under treatment with pilocarpine. During the operation, a combined extraction, the capsule gave way when grasped with the forceps. The nucleus was expressed and there did not seem to be much fluid cortex, the pupil remaining black. During convalescence the anterior chamber remained shallow and the tension low; a choroidal detachment was present. Later the tension rose to 55mm. with a deep anterior chamber and steamy cornea. After a Lagrange operation the tension remained normal but there was considerable iridocyclitis with corneal opacity. The pupil was adherent to thickened capsule attached to the wound. In the second case the tension was also raised and reduced by treatment on two occasions before the performance of an iridectomy. Later the cataract was successfully extracted with blunt capsule forceps in the capsule. Recovery was uneventful and normal vision was regained.

In commenting on these two cases, Knapp considers that they are suggestive of a low-grade uveitis in which the increased tension is the most striking feature. He does not agree with Gifford in thinking that the liquid cortex is the toxic source of the disturbance but inclines to the opinion that the displaced nucleus must be the cause of some mechanical irritation of the ciliary processes and thus produce the glaucoma. Extraction in the capsule is undoubtedly the operation of choice in these cases, but is technically by no means easy of performance owing to the condition of the capsule.

In the ensuing discussion in which opinions on the toxicity or otherwise of the Morgagnian fluid were illustrated by a number of cases, all were agreed as to the importance of not allowing a cataract to advance to the Morgagnian condition, and as to the advisability of intracapsular extraction in such cases.

E.E.H.

(4) **Hamburger, Carl (Berlin).—The new treatment of glaucoma. (Zu der neuen Glaukombehandlung.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXII, S. 47, 1924.

(4) **Hamburger** points out that mydriasis and miosis in the treatment of glaucoma are held to have actions diametrically opposed to each other, and yet adrenaline by subconjunctival injection, the most powerful mydriatic known, can effect the following:

(1) Reduce the tension of a normal eye to a "soup-like" softness for hours or even for days; (2) favourably influence glaucomatous eyes, an influence which can be prolonged by repeated injections at regular intervals; (3) reduce the tension in the fellow uninjected eye, thus settling the question whether an irritation can be transferred from one eye to another; (4) that its hypotonic power can be considerably improved by a combination with eserine. This is especially useful in cases where eserine alone has proved impotent. That is to say that the most powerful mydriatic has its hypotonic power enhanced by combination with a miotic. The injections are made as follow:

A few drops of a 2 per cent. solution of holocaine are instilled with sufficient adrenaline to contract the vessels. A fold of conjunctiva is raised on the temporal side and about 0.3 c.cm. of 1:1,000 adrenaline injected. After about five minutes the pupil begins to dilate, always eccentrically, and after about fifteen minutes is dilated *ad maximum*, sometimes the iris almost disappears. In iritis synechiae are torn away, often with considerable pain, even when they have failed to yield to atropine and scopolamine. The mydriasis is so powerful that it persists even when the anterior chamber is opened and the aqueous escapes. In most cases of glaucoma the tension falls, sometimes after a slight preliminary

rise. In a few cases an acute glaucoma has supervened. Seven such have been described but all were cured by the use of eserine without operation. This complication can generally be avoided by instilling eserine energetically about an hour after the injection, just as one would proceed after a diagnostic instillation of homatropine, but more frequently. The softening caused by adrenaline is easily explained. It constricts the afferent small vessels, because adrenaline is the strongest stimulant for the sympathetic vaso-constrictors. The cavernous sinuses of the choroid are compressed like the squeezing of a sponge, since the massive uveal water cushion that encircles the whole eye is relatively poor in capillaries.

It is more difficult to account for the hypotonic action of eserine, since the pupil contracts, the iris vessels dilate and become engorged, and even the ciliary body is hyperaemic. This can be seen with the naked eye in an albino rabbit. The secretory action of the ciliary body is stimulated and the albumen content of the aqueous rises. We have increased supply of blood, increased metabolism, and yet the tension falls, a physiological paradox! The explanation lies in the fact that the circulation of the iris is quite distinct from that of the rest of the uvea, the former is supplied by the long, the latter by the short ciliary arteries. The iris in consequence does not share in the affections of the choroid and ciliary body, and the converse is equally true. The two circulations are antagonistic to each other, one compensates the other. Eserine causes hyperaemia of the anterior part of the eye, and as a compensation the posterior part is ischaemic. The combined effect of adrenaline and eserine is due to the fact that adrenaline constricts the capillaries of the eye and so squeezes out the choroidal water cushion, whereas eserine draws the blood that still remains in the eye forward, and smooths out the iris folds, so increasing the absorbing surface at a time when the adrenaline mydriasis is passing off.

Glaucoma is due to a variety of conditions and naturally adrenaline treatment like all others occasionally fails. Fisher calls glaucoma "ocular dropsy" and draws attention to the capability of the vitreous to become swollen, and it is possible that the acute forms of glaucoma are due to swelling of the vitreous. This may explain the fact that adrenaline treatment is ineffective in acute glaucoma. Finally, the indications for treatment in glaucoma are: first miotics, then a combination of eserine drops with injections of adrenaline, and finally, only when this fails, operation. It is necessary in chronic glaucoma to repeat the injections at about three-weekly intervals.

T. HARRISON BUTLER.

(5) **Agnantis, C.—Modifications of the visual field in secondary glaucoma. (Les modifications du champ visuel dans le glaucome secondaire.)** *Ann. d'Ocul.*, T. CLXII, p. 597, 1925.

(5) **Agnantis** describes in detail, with a number of charts of fields of vision, a series of eighteen cases of secondary glaucoma. He refers to the book by F. Lagrange "Glaucome et Hypotonie" in which glaucoma is divided into "glaucome vrai"—referring to primary glaucoma—and "glaucome faux"—secondary glaucoma, and in which Lagrange denies the existence of typical field contraction in the second group (secondary glaucoma).

As a result of the cases described, he claims that in every case in which for an unknown reason (*i.e.*, primary glaucoma) or for a known reason (*i.e.*, secondary glaucoma) the ocular tension is raised for a sufficient length of time, contraction of the visual field results. This is due mainly to ischaemia of the peripheral retina from pressure. Failure of this general statement occurs occasionally in both primary and secondary glaucoma when the rise of tension is only temporary and occasional. The nasal field suffers particularly owing to (1) the greater length of the vessels to the part of the retina corresponding with this field; (2) the early affection of the temporal half of the disc by cupping. Agnantis concludes by pointing out that nine of the eighteen cases show conclusively that nasal loss of field is just as typical in chronic secondary glaucoma as in primary glaucoma. Is it not possible, he says, that primary glaucoma owes its development to some lesion as yet not recognized, and that it is secondary to such a lesion, so far unknown, but perhaps to be discovered in the future?

HUMPHREY NEAME.

(6) **Meesmann, A. (Berlin). — Contributions to the physical chemistry of the modification of the intraocular fluid under normal and pathological conditions, particularly in glaucoma. (Beiträge zur physikalischen Chemie der intraokularen Flüssigkeitswechsels unter normalen und pathologischen Verhältnissen insbesondere beim Glaukom.)** *Arch. f. Augenheilk.*, Bd. XCVII, December, 1925.

(6) As the details of this research are too long and complicated to be abstracted, the interested reader is referred to the original for them. The following is **Meesmann**'s summary:

"(1) Glaucoma is not to be explained by differences in the osmotic pressure of blood. The membranes separating blood and aqueous are wholly permeable to crystalloids. Hence differences in the crystalloid osmotic concentration of blood cannot lead to prolonged change of tension inside the eye. But in regard to colloid osmotic pressure there is such a possibility. Nevertheless, according to

Serr's researches, such deviations from the normal of colloid pressure do not occur in the blood of the glaucoma patient. Neither does the view that osmotic pressure accounts for variations of intraocular tension derive any support from Donnan's formula for osmotic equilibrium.

(2) The significance of the actual reaction of the aqueous to the intraocular tension is again stressed and confirmed by new measurements. If the alkalinity of the aqueous rises above the normal there is corresponding increase of tension, and fall of alkalinity below normal regularly leads to fall of tension. The physico-chemical processes underlying these relations are the swelling and detumescence of the vitreous and the lens.

(3) As a rule a corresponding change in the blood could be shown to be the cause of the actual change of reaction of the aqueous, *e.g.*, an acidosis of the blood in diabetic coma and in the later months of pregnancy, an alkalosis in primary glaucoma. The alkalosis is a permanent condition in chronic glaucoma whereas in acute glaucoma it appears to be transient. On the contrary secondary glaucoma is wholly dependent on local changes.

(4) The essence of primary glaucoma is an increased swelling of the contents of the eye, caused by alkalosis of the blood and consequently of the aqueous. Hence in grave diabetes and the later months of gestation primary glaucoma is improbable, a surmise which is well supported by clinical facts. Similarly the relative infrequency of primary glaucoma in high myopes is intelligible on account of the degeneration of the vitreous and its consequent diminished capacity to swell.

(5) The individual symptoms of primary glaucoma and the effectiveness and failure of different therapeutic measures are easily explained by the alkalosis. The mydriasis—hitherto not satisfactorily explained by any theory—is, according to the researches of Wieland and Schoen, also to be regarded as a consequence of alkalosis of the blood, *i.e.*, a paralysis of the miotic centre through the decreased CO_2-tension of the blood."

D. V. GIRI.

(7) **Bailliart and Magitot.—The behaviour of the circulation in glaucoma. (Le régime circulatoire du glaucome.)** *Ann. d'Ocul.*, T. CLXII, p. 729, 1925.

(7) According to **Bailliart** and **Magitot** the tension of the eye is found to vary normally with the systemic blood pressure. In a large proportion of cases of chronic glaucoma, the general blood pressure is raised. In certain cardio-renal cases, however, the ocular tension is low in spite of enormously high blood pressure. In such cases, the sclerosis of small vessels with some degree of added arterial spasm reduces the volume of blood in the eye and so the tension. The circulatory trouble in glaucoma is one rather

of the exit than the entry of blood into the eye. In the normal eye the blood pressure in the central artery of the retina and its main branches is as follows: minimum 30 to 35 mm. of mercury, maximum 70 mm. The venous pressure is approximately equal to the ocular tension. Seidel is quoted as finding that the pressure in the anterior ciliary artery perforating branches is equal to 30 to 35 mm. minimum and 55 to 75 mm. maximum. (Bailliart and Magitot find the choroidal arterial pressure equal to the retinal arterial pressure, by observation of the circulation in an atrophic patch in the choroid.) Seidel in a similar manner found the pressure in the anterior ciliary vein equal to 10 to 14 mm. of mercury. Bailliart and Magitot consider that the choroidal venous pressure is about equal to the intraocular pressure, for, if the choroidal venous pressure were higher than this in the anterior ciliary perforating veins of the eye, these would become distended and so automatically lower the pressure. The walls of the intraocular veins are supported by the intraocular tension, whereas the anterior ciliary veins outside the eye are not supported. There is a corresponding pressure in the arteries of the choroid and in the retina.

In raised intraocular tension the minimal retinal arterial pressure is almost always raised. At the same time, the venous retinal pressure must rise to avoid obliteration, and therefore probably equals approximately the tension of the eye, as in normal conditions. In glaucoma the anterior ciliary and subconjunctival veins show raised pressure by their frequent marked dilatation and by their rapid filling from the limbus peripherally, after compression by the eyelid. Within the eye the raised tension prevents marked dilatation of veins which have raised blood pressure. Further, the dilatation of veins on the iris surface in long-standing glaucoma, and especially in cases with central retinal vein thrombosis, signifies raised intravenous pressure in the ciliary vein.

To summarize, in raised intraocular tension, there is a moderate increase of arterial blood pressure and often a marked increase of venous blood pressure. The ocular tension is not affected by obstruction of the central artery of the retina, nor, at least at first, by obstruction of the central vein. Is this true of the choroid? This is considered the kernel of the situation. It is probable that obstruction to the choroidal circulation has a definite effect, as the volume of the choroidal circulation is infinitely greater than that of the retina. It is possible that the rise in arterial pressure is compensatory and secondary to slowing of the venous circulation. The following observations are tabulated:

(*a*) Obstruction of the vortex veins causes marked hypertension, as also does obstruction of the anterior ciliary veins.

(*b*) In animals bled to death, perfusion restores the tension of the eye to normal (Weiss, C.).

(*c*) Carotid compression reduces the tension.

(*d*) The introduction of adrenaline into the retrobulbar tissues, and to a less degree into the subconjunctival tissues, reduces the tension (Fromaget).

(*e*) On the other hand, irritants, sodium chloride, mercuric chloride, cause a rise of tension owing to the resulting vaso-dilatation (Wessely).

(*f*) Amyl nitrite by causing capillary dilatation also raises the tension (Wessely, Leplat, Bailliart, and Bollack).

(*g*) Contusion of the eye by resulting hyperaemia of the choroid, also causes a rise in tension, not only in the injured eye, but also in the other (Leplat).

(*h*) Section of sympathetic vasomotor nerves raises the tension, and stimulation of these nerves lowers the tension, concurrently with changes in the state of the vessels (Bailliart and Magitot).

Several cases are reported in detail of primary glaucoma in which pathological examination of the eye was possible during the condition of glaucoma owing to the supervention of death from some intercurrent cause, such as pneumonia or accident. Pathological examination showed various signs of inflammation in the eye, with perivascular inflammation and sclerosis of episcleral veins and vessels of iris and choroid. Reference is made to obstruction by experimental ligature of the vortex veins.

Conclusion.—Glaucoma may, therefore, be caused by venous obstruction from phlebitis or by overgrowth of the vein wall occurring particularly in the ciliary circle. The result of this venous obstruction is resistance to the emptying of the capillaries. This is of greater importance for the choroid than for the retina as the retinal circulation is a mere fraction in volume compared with that of the choroid. Vaso-dilatation follows, presumably owing to nerve impulses, in order to overcome the venous obstacle. As a result of this, the capillary reservoir dilates and ocular hypertension supervenes. Similar hypertension may occur in general arterio-sclerosis with general rise of blood pressure. When the ocular tension becomes equal to the arterial diastolic pressure, arterial pulsation appears in the retina. The observation of arterial pulsation is of practical importance. When the ocular tension becomes equal to the diastolic arterial pressure of the central artery, an indication for operation is obtained. The central artery blood pressure in the normal eye is approximately equal to half the blood pressure in the humeral artery.

HUMPHREY NEAME.

(8) **Gros, H. B. (Boulogne-sur-mer).—Glaucoma following capsular adhesions. (*Glaucome consecutif à des enclavements capsulaires.*)** ***La Clin. Ophtal.,*** December, 1925.

(8) In the case recorded by Gros glaucoma resulted from fine capsulo-corneal adhesions and when these were divided the tension returned to normal. The eye had been injured by the explosion of a detonator, lenses wounded, cataract successfully extracted in the eye under consideration, unsuccessfully in the other.

ERNEST THOMSON.

II.—DISEASE OF RETINA

(1) **Shoji, Y. (Okoyama, Japan).—Congenital unilateral non-syphilitic retinitis pigmentosa. (Rétinite pigmentaire congénitale unilatérale non-syphilitique.)** ***Arch. d'Ophtal.,*** July, 1926.

(1) Retinitis pigmentosa congenitalis is almost invariably bilateral, and although certain observers have refused to entertain the idea of uniocular examples of the disease a number of cases have been reported, to which reference is made in this paper.

Shoji has now published complete notes of a case in which the evidence appears to be unequivocal.

The patient, a healthy Japanese youth, came under observation when aged 13 years. For about a year he had noticed that in the dusk the vision of the left eye was feebler than that of his right: Central vision, however, was R. 0.2; L. 0.8. Fields of vision: R. normal; L. concentrically narrowed for white, red, and green. Ophthalmoscopic examination: R. fundus normal; L. numerous small stellate pigment deposits in the retina peripherally. Charts of the fields and drawings of the right and left fundi at this date are given. The Wassermann reaction was negative. Family history: consanguinity in the grandparents. Parents healthy, no miscarriages.

After the lapse of ten years during which no treatment had been adopted, the patient reported increased defect of his left eye, and loss of distant vision. Examination at this date showed central vision in the R.=0.5 with -2D. sph.; in the L.=0.9 with -0.75D. sph.

The field of the R. normal for white and colours; the field of the L. concentrically contracted to less than 10° all round. Light-sense tested with Foerster's photometer (after 15 mins. dark adaptation) R. 2 mm.; L. 22 mm. With Nagel's adaptometer dark adaptation was normal in the right, very feeble in the left, as shown by curves in diagram. Colour-sense in central field was normal in R. and L.

Ophthalmoscopically: The right fundus was healthy; the left showed diminution of the retinal arteries; retina uniformly dull; much black stellate pigment deposit beyond the immediate neighbourhood of the disc and macula. On the anterior lens capsule there were about thirty small brown dots thought to be persistent remains of the pupillary membrane.

The Wassermann reaction was negative throughout.

J. B. LAWFORD.

(2) **Coppez, H., and Danis, M. (Brussels).—Senile macular exudative retinitis and retinitis circinata. (Rétinite exsudative maculaire sénile et rétinite circinée.)** *Arch. d'Ophtal.*, August, 1926.

(2) In this additional contribution concerning senile exudative retinitis **Coppez** and **Danis** devote especial attention to the relationship between this disorder and circinate retinitis, to which they drew attention in 1923. Several observers have written on the subject since that date. Batten (*Trans. Ophthal. Soc., U.K.*, 1923) published a series of drawings of macular lesions in arteriosclerosis, some of which Coppez and Danis believe to be examples of senile macular exudative retinitis. Two cases showed lesions characteristic of circinate retinitis. Feingold (*Trans. Amer. Ophthal. Soc.*, 1924) reported seven cases under the title proposed by the Belgian authors in 1922. He holds that senile exudative retinitis should be clearly differentiated from retinitis circinata and from angeioid streaks in the retina. Junius and Kuhnt in an illustrated monograph on cases designated by them "disciform degeneration of the macula lutea" report ten cases, three of which presented the signs of circinate retinitis.

Such statistics as exist indicate that circinate retinitis is a rare disease. Fuchs reports eleven cases in 70,000 patients; de Wecker fifteen in 150,000; Silex three in 23,000. It occurs in persons over 50 years of age and is characterized by slow and progressive failure of sight. Ophthalmoscopically, there are two chief signs: (1) an elliptical white corona, its long axis horizontal, encircling the macular region; it is composed of small rounded white spots posterior to the vessels, and isolated or confluent. The corona is usually broken on the temporal side. (2) A macular lesion which is, however, not constant; it consists of a zone of greyish or yellowish discolouration occupying the macular area; it varies considerably in size and in the definition of its borders, and generally shows central haemorrhage or pigmentation. It does not reach the corona. The retinal vessels seldom show any alteration but in some instances are tortuous. Anatomical examination of circinate retinitis has been made once only, by von Ammon. He reported that the white deposits in the retina

were external to the inner granule layer and that where the exudation was situated the rods and cones and the outer granules had disappeared; the other layers of the retina were thickened, as were Müller's fibres.

de Wecker and von Ammon believe that the white deposits are the remnants of former haemorrhage; Fuchs disagrees and considers that they are analogous to the exudation in albuminuric retinal lesions and are composed of transuded albumen.

There are numerous analogies between senile exudative retinitis and circinate retinitis. Both arise without appreciable cause in elderly people; their evolution is slow and progressive; the vascular lesions are discrete; haemorrhages are frequent; the seat of the lesions is the posterior pole of the eye.

In this paper Coppez and Danis publish notes, with coloured plates, of two cases of senile exudative macular retinitis accompanied by the usual appearances of circinate retinitis. One of these examples was seen on one occasion only; the other has been under observation for ten years, and the drawings show the fundus changes at an interval of two and a half years. Examination of the latter case by means of the binocular ophthalmoscope and in red-free light showed that the haemorrhages are situated in the external layers of the retina or in the choroido-retinal space; the white spots are internal to the haemorrhages but are external to the retinal vessels; in the area of the macular exudation there are small deep blood extravasations in addition to pigmented spots; the nerve fibres are probably atrophied as their fibrillar arrangement is invisible in red-free light.

J. B. LAWFORD.

(3) **Velter, E., and Blum, J. (Paris).—A case of ocular tuberculosis originating in the retina.) (Sur un cas de tuberculose oculaire à point de départ rétinien).** *Arch. d'Ophtal.*, March, 1926.

(3) The case here reported was under clinical observation for a period of three or four months: the eye was then excised and submitted to histological examination. Although the evidence is not wholly without flaw there seems good reason for Velter and Blum's contention that the tuberculous lesion began in the retina. Accompanying the report are to be found one drawing of the ophthalmoscopic changes and two micro-photographs of the pathological appearances.

The patient, a male, aged 36 years, had pulmonary and other lesions considered tuberculous in nature. When first seen the left eye exhibited no signs of disease in the anterior part; the media were clear and the fundus easily illuminated. The papilla was hyperaemic, an elongated patch beginning close to the uninvolved

margins of the disc, extended across and beyond the macular region, bounded above and below by the temporal retinal vessels. It was grey in colour becoming intensely white at its periphery. There were also small separate rounded nodules scattered along the temporal retinal vessels especially the lower branches, similar in colour to the central plaque. No pigmentation and no peripheral changes were discovered. The field of vision showed a corresponding area of blindness. Tonometric tension was 15.

Three months later the condition had undergone great change. There were photophobia, lacrymation, and severe pain; the eyeball was extremely tender; the lids and conjunctiva were swollen. The cornea was clear, the anterior chamber abolished, the iris dull, bulged, and the pupil filled with exudation. Tension by the tonometer was 40.

Sections of the globe showed follicular caseating tuberculous lesions involving all the posterior chamber; only the cornea and anterior chamber had escaped the bacillary infection. There was massive invasion of the retina which was completely detached and almost wholly destroyed except anteriorly near the ciliary processes: the appearances were characteristically tuberculous. The choroid showed chronic inflammatory fibro-vascular lesions attaining their maximum at the posterior pole of the eye. In the portion of the choroid thus altered no trace of follicular tuberculous lesions was found; it was covered by a degenerating fibrinous layer containing scattered giant cells. In the anterior segment of the globe were recent lesions of tuberculous character in the choroid. Neither the ciliary processes nor the iris were obviously tuberculous: the latter was very vascular and thickened.

In cases of ocular tuberculosis in which the initial lesion is choroidal, invasion of the retina is frequent and often considerable, but the concomitant lesions of the uveal tract prove the primary seat of disease to be in that tissue. In the present case the order appears to be reversed; the retina shows a massive invasion by tubercle while the choroidal lesions are those of simple inflammation except in the anterior part where the lesions are recent but characteristically tuberculous.

J. B. LAWFORD.

(4) **Lavrand, H., and Desprets, H. (Lille).—Pigmentary choroido-retinitis following a latent chronic anterior ethmoiditis. (Chorio-rétinite pigmentaire consécutive a une ethmoidite antérieure chronique latente.)** *La Clin. Ophtal.*, August, 1926.

(4) **Lavrand** and **Desprets** relate in detail the case of a woman, aged 35 years, who suffered from asthenopia, headache, and loss of vision in one eye, in whose fundus were found choroido-retinal pigmented lesions of various ages. The patient was in various ways not in a healthy condition, but there was no direct

evidence as to the aetiology. Basing themselves on previous clinical experience and in spite of the absence of symptoms of frontal, maxillary, or sphenoidal sinusitis it was decided to explore the anterior ethmoidal cells. A hitherto undetected osteitis was found on both sides. Active treatment of this resulted in cessation of the headaches and the asthenopia. Visual acuity rose from barely 1/20th to 1/4th or 1/3rd.

ERNEST THOMSON.

(5) **Friedenwald, Dr. H., and Friedenwald, Dr. J. S. (Maryland). —Globular masses on the pupillary margin in acute circumscribed chorioretinitis. Clinical and pathological study.** *Arch. of Ophthal.*, March, 1926.

(5) **Harry** and **Jonas Friedenwald** bring forward some interesting evidence in support of the theory that keratic precipitates may, in some cases, be derived from a focus of choroidal infection, without there being an iritis or cyclitis. Moreover, some of the cells passing forwards to the keratic precipitates may be deposited on other structures en route. Thus we have (1) the formation of pupillary globules; (2) globules hanging on to the fibres of the vitreous; (3) cells on the surface of the ciliary body without underlying inflammation; and (4) perivascular stripes in the retina. The evidence comprises the cases in which there were globular masses on the margin of the pupil. In four of these a patch of exudative choroiditis was found, in five there were extensive vitreous opacities, and the last was a case of iritis. In one case of acute exudative central choroiditis the eye was removed on account of the possibility of the presence of a growth and a full pathological examination was made. The presence of the choroiditis was confirmed and there was necrosis of the overlying retina. The interesting feature, however, was the occurrence of small lymphocytes, large cells with oval eccentric nuclei, and vacuolated protoplasm in positions far removed from the original focus of disease. Many of these cells were laden with the products of phagocytosis. The authors therefore consider that some of them at any rate are cells in process of being carried away by the lymph currents and therefore that the presence of keratic precipitates and of globular masses in the iris may be associated with a lesion much further back than the ciliary body.

F. A. WILLIAMSON-NOBLE.

(6) **Bettremieux. — Retinal detachment and ocular tonus. (Decollement rétinienne et ophtalmotonus.)** *La Clin. Ophtal.*, August, 1926.

(6) **Bettremieux** refers to a communication by Weekers in which the latter had pointed out, apparently as a new point, that

in detachment of the retina there is sometimes a rise of tension at the beginning. Bettremieux states that it was recognized some time ago by himself, Boucheron, and Dransart that in detachment there are two periods of ocular tension, one at the commencement when the tension is raised, and a later period of lowered tension. The early hypertension is sometimes appreciable by the finger but frequently is only manifested in the course of an iridectomy by the occurrence of a jet of liquid analogous to that in a case of moderate glaucoma. These slight increases of tension may occur several times in the early days of a retinal detachment when fresh exudations are occurring. According to Boucheron the indication is to operate early when there is a certain degree of hypertonus, and the author has come to the same conclusion. But Bettremieux prefers his own operation to iridectomy. This operation is that of non-perforating pericorneal sclerectomy. The reader will find abstracts in *The Ophthalmoscope* for 1908, pp. 400 and 818, and probably elsewhere either in that journal or its successor, dealing with the Bettremieux operation.

ERNEST THOMSON.

III—MISCELLANEOUS

(1) **Coppez, Henri (Brussels).—Examination of the aqueous humour for the presence of iron, in relation to the diagnosis of intraocular foreign bodies. (Sur la recherche du fer dans l'humeur aqueuse considérée au point de vue du diagnostic des corps étrangers intra-oculaires.)** *Arch. d'Ophtal.*, October, 1926.

(1) Despite the various aids to diagnosis commonly employed (giant magnet, radiography, etc.), the nature and even the presence of minute foreign bodies within the eyeball occasionally escape detection. In some instances, five of which **Coppez** quotes from published records, late evidence of the foreign body is forthcoming in the development of siderosis.

It is, for several reasons, desirable to obtain positive evidence of an intraocular foreign body, metallic in nature, in cases in which the means cited above have failed: among them is the fact that the majority of such cases being industrial accidents, lack of definite information might prevent the artisan obtaining compensation to which he was entitled.

In 1921, Coppez had recourse to chemical analysis of the aqueous, and reported his first case, at the time, to the Belgian Ophthalmological Society. In the present communication he gives detailed notes of the original and of three recent cases. In all four the presence of iron in the aqueous was indubitably shown by chemical analysis, after a negative result by other methods of examination.

In one case in which the eyeball had to be removed for secondary glaucoma a splinter of iron was found embedded in the sclera at the posterior pole.

The procedure to be followed should be studied in the Coppez paper: meticulous care is necessary to avoid all possibility of error and to exclude that which might arise from the presence of haemoglobin in the aqueous. The analysis should be entrusted to an expert analytical chemist.

The subject of this communication is important and the author's facts and statements are interesting: in his concluding sentences he says:—"I feel able to state from these observations that the examination of the aqueous humour for the presence of iron is a good diagnostic procedure. It is especially noteworthy by reason of its value at a date so distant from the accident that the usual methods have become uncertain or completely ineffective. I advise its adoption therefore in all cases of traumatism in which the abnormal development of lesions, or the appearance of signs of siderosis suggest the presence of a foreign body previously unsuspected."

J. B. LAWFORD.

(2) **van Lint (Brussels). — Palpebral Akinesia. (L'Akinésie palpébrale.)** *Arch. d'Ophtal.*, December, 1926.

(2) In 1914 **van Lint** published an article in *Ann. d'Ocul.*, Vol. CLI, 1914, on "Transient palpebral paralysis during operation for cataract," in which he stressed the danger from spasmodic contraction of the lids during extraction of cataract and showed how this danger could be obviated by the injection of novocaine-adrenaline in the course of the facial nerve fibres before they penetrate the orbicularis. While control of the lids is helpful in extracapsular extraction it is almost indispensable in intracapsular extraction in which the larger wound facilitates loss of vitreous.

Since that time palpebral akinesia (the term given by Rochat) has been widely adopted by ophthalmic surgeons. But though the majority employ this method there are still some who have not adopted it for various reasons. The present communication is specially addressed to these latter in the hope of convincing them that with an adequate technique happy results are constant. The reviewer believes that comparatively few surgeons in this country have adopted this practice and for that reason gives van Lint's directions almost in full.

van Lint uses a hypodermic syringe holding 4 c.c. with a strong steel needle 3 cm. in length. The solution injected (4 c.c.) is novocaine 2 per cent. with 2 to 3 drops of adrenaline added. Anaesthesia of the surface of the eyeball is begun and immediately after this the injections are made, *i.e.*, about 15 minutes before operative measures.

In order that the injection may be successful it is necessary to prevent motor impulses from the centres reaching the orbicularis, by impregnating the terminal filaments of the facial nerve with novocaine. These filaments reach the muscle, grouped on the bone of the external and inferior borders of the orbit. The injection must therefore be made in this position and to the requisite depth. Iodine is applied to the skin: the needle is passed into the skin perpendicularly and down to the bone, at the intersection of a horizontal line, passing 0.5 cm. below the inferior edge of the orbit, and a vertical line passing 0.5 cm. outside the external margin of the orbit. One drop is injected: the needle is partially withdrawn and turned in the direction of the nose along the horizontal line already designated, and at each half centimetre point an injection is made, always feeling the point of the needle on the bone, until the inner end of the lower lid is reached. About 1.5 c.c. of the solution is injected during this procedure.

For the injection at the lower half of the temporal border of the orbit, the needle is not completely withdrawn from the first site but its point is directed upwards near the bone and half a cm. from the orbital margin. One c.c. is injected here by the same intermittent manoeuvre as before. The needle is then withdrawn. It is re-inserted from above downwards at a point on the frontal ridge at its intersection with a vertical line along the outer margin of the orbit. The remaining 1.5 c.c. of the solution is injected here. This is in brief the technique recommended, and the author says: "the results are constant, sometimes greater, sometimes less, but always sufficient to render the patient incapable of closing his lids forcibly. If the injections are deep and not excessive in amount no oedema of the lids is induced.

van Lint has advocated and practised this method consistently and is convinced of its great utility. He says, epigrammatically, "Celui qui opère la cataracte sans l'akinésie palpébrale a la sensation de faire de l'acrobatie, de marcher en équilibre sur une corde tendue. Celui qui fait l'akinésie marche d'un pas assuré sur la terre ferme."

J. B. LAWFORD.

BOOK NOTICES

Transactions of the American Ophthalmological Society. Vol. XXIV, pp. 397, Philadelphia, 1926.

The sixty-second annual meeting of the American Ophthalmological Society was held at Hot Springs, Va., in June, 1926; the present volume, in addition to the papers read and the discussion thereon, includes some features of more than domestic interest in

the minutes of the proceedings. The reports of several special committees were presented. The committee appointed to investigate and revise the classification of certain retinal conditions made a final report recommending that the term "retinoblastoma be hereafter used to designate the tumours heretofore designated as glioma of the retina, gliosarcoma, medullary cancer of the retina, neuro-epithelioma retinae, etc." This term was suggested by Verhoeff as fitting in with every known fact regarding such tumours. The committee made no recommendation as to the nomenclature of the chronic degenerative condition known as gliosis retinae (v. Hippel's disease) as they did not consider that the present state of our knowledge warranted them in deciding on the pathology of this disease.

In discussing the appointment of a committee to consider questions relating to optometry Dr. Edward Jackson pointed out that in America there were three bodies that might be regarded as nationally representing ophthalmology—the Section on Ophthalmology of the American Medical Association, the American Academy of Ophthalmology and Oto-Laryngology, and the American Ophthalmological Society. He considered that America even more than Great Britain was in need of some representative body such as the Council of British Ophthalmologists. No immediate steps, however, were taken to constitute such a body.

The proceedings closed with a pious resolution declaring that the Society heartily approved of legislation which will tend to lessen hereditary blindness. We propose to give abstracts of the more important papers at a later date.

Physical Therapy in Diseases of the Eye, Ear, Nose, and Throat; with chapters on some borderline affections. By HOLLENDER AND COTTLE. 8vo., pp. 17 and 307, with 81 illustrations. London: J. & A. Churchill, 1926. Price 21s. net.

This book aims at gathering together the facts concerning the treatment of certain affections of the eye, ear, nose, and throat by means of electro-physical agents. The first section deals with these agents, which are enumerated as follows:—The galvanic current, high frequency currents-diathermy, the sinusoidal current, radiant heat-light, ultra-violet light and X-rays. In the treatment of ophthalmic conditions, which forms the second section, short accounts of the treatment of diseases of the lids and conjunctiva and lacrymal apparatus, the cornea, uveal tract and retina, such optic nerve conditions as atrophy and neuritis, vitreous opacities, cataract and glaucoma are given.

We gather that radiant heat-light is, as an adjuvant, valuable in the treatment of such diseases as hordeolum, eczema of the skin of the lids, acute simple conjunctivitis; and that ultra-violet light is of

use in the treatment of chronic conjunctivitis, and "by compression" in cases of chalazion.

The authors hold that "no medicinal treatment is effectual against cataract, but the electrical currents hold out some hope in selected cases." As cataract from electric shock is well known, we suppose that the treatment of cataract by electricity must be undertaken with caution; treatment is said to be prolonged and the results "successful in some incipient cases." In this journal we have expressed our opinion that the "cure of cataract without operation" is an unjustifiable assumption in the present state of our knowledge, and we will leave the subject at that.

The rest of the book does not immediately concern ophthalmic surgeons who practise in this country.

CORRESPONDENCE

PROPTOSIS IN A NEW BORN CHILD

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—With regard to the references to proptosis in the March number of the *Brit. Jl. of Ophthal.*, p. 141 the following cases should be added to those mentioned.

Berger (Maladies des Yeux, p. 258), quotes a case, seen by Steinheim, in which the ocular muscles were torn by forceps at birth. Purulent keratitis followed with perforation of the cornea. The child died from meningitis in eight days and at the autopsy a fracture of the vault of the orbit was discovered.

Hoffmann is quoted in the same book with respect to an infant delivered by instruments, in which the eye was completely outside the orbit and attached to it only by a fragment. In another case Spaeth, of Vienna, saw a child in which after the use of forceps both eyes were torn.

Fage, of Amiens, refers to a case of Bock's (*Central. für prakt. Augenheilk.*, 1902), in which a face presentation was mistaken for a breech, and the accoucheur passed his finger into the orbit thinking it was the anus, intending to pull the foetus. A dislocated eye resulted followed by panophthalmitis.

In Fage's case the left eye of a female child was completely displaced from the socket after a normal confinement. The day preceding the birth the woman had been struck severely on the lower part of the abdomen by the shaft of a cart. The eye was replaced in the orbit and the lids sewn together. The eye became

leucomatous but otherwise the result was satisfactory. Dislocation of the eye is known by several names: proptosis, displacement, exophthalmos. Personally I prefer to keep dislocation for cases which are traumatic in origin and proptosis for those associated with myopia, goitre, etc. Exophthalmos is the result.

Yours faithfully,

W. M. BEAUMONT.

BATH.

OBITUARY

ALEXANDER DUANE, M.D., D.Sc.

THE fact of Dr. Duane's death was briefly noticed in our pages in Vol. X, and an obituary was promised for some future occasion. The present note is an abstract of the sympathetic notice contained in the *Trans. Amer. Ophthal. Soc.*, Vol. 24, which was contributed by Duane's old friend, John E. Weeks.

Dr. Duane was born in Malone, N.Y. in 1858, the son of General James Duane. He died in New York, of meningitis, on June 10th, 1926.

He was educated at Union College and qualified in Arts in 1878, obtaining his M.D. in 1881. After holding a resident appointment at the New York Hospital, he began to practise in New York City in 1884. Four years later he moved to Norfolk, Virginia, where he stayed for a couple of years and then returned to New York, where he practised for the rest of his life. For many years he was associated with the late Herman Knapp. He was a voluminous writer, and besides some 70 papers contributed to various journals, he supplied the medical terms for Webster's International Dictionary, and those of ophthalmology for Foster's Encyclopaedic Dictionary of Medicine. His Student's Medical Dictionary was published in 1893, and ran through four editions. His *magnum opus* was undoubtedly the translation of Fuchs's well-known textbook, which has reached its seventh edition. In 1897, his "Motor Anomalies of the Eye" appeared. Besides all this, he contributed chapters to most of the American ophthalmological textbooks of his time.

Duane served in the Spanish-American War as Lieutenant, U.S.N. and in the Great War as signal officer, U.S.S., "Granite State." He was the author of "Rules for Signalling on Land and Sea" (1899, second edition 1901).

In 1919 he received the honorary degree of D.Sc. from his old college. He became a member of the American Ophthalmological

Society in 1902, received the Howe medal in 1923, and was President of the Society in 1924.

Duane was specially interested in physiological optics and was a recognized authority on the movement of the eyes and their motor anomalies.

NOTES

Central London Ophthalmic Hospital Dinner

THE Annual Dinner of past and present Students of the Central London Ophthalmic Hospital was held at the Langham Hotel on March 11; Mr. Ernest Clarke, C.V.O., in the chair.

The toast of the hospital and school was proposed by the Chairman, and replied to by the Chairman of the Board of Management. Mr. Williamson-Noble, as Dean of the School, also replied. The health of the visitors was proposed by Mr. Mayou, and replied to by Sir John Parsons. Finally, Mr. A. Harold Levy proposed the health of the Chairman.

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Death

AS we go to press we hear with much regret of the death of Mr. A. Stanford Morton. We hope to publish an obituary notice in a later number.

FUTURE ARRANGEMENTS

1927

May 9—12.—Congress of the Société française d'Ophtalmologie, at Paris.

May 13.—Royal Society of Medicine, Section of Ophthalmology.

June 10.—Royal Society of Medicine, Section of Ophthalmology (Annual).

July 7-9.—Annual Meeting of the Oxford Ophthalmological Congress at Oxford.

July 20-22.—The British Medical Association, Section of Ophthalmology, at Edinburgh.